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EARLY DIAGNOSIS

OF SOME SERIOUS

DISEASES OF THE NERVOUS SYSTEM:

ITS IMPORTANCE AND FEASIBILITY.

BY

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EARLY DIAGNOSIS OF SOME SERIOUS DISEASES OF THE NERVOUS SYSTEM; ITS IMPORTANCE AND FEASIBILITY.¹

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THERE are several serious diseases of the nervous system, and others which, at an early stage present chiefly nervous symptoms, that are held to be incurable, or nearly so.

One reason for this dismal prognosis is that in the present state of medicine the diagnosis of these affections is usually only made when organic changes are far advanced, or, in the case of epilepsy, when the functional disturbance has become firmly fixed as a habit of the nervous system. Such cases almost never reach nervous specialists on the first appearance of symptoms; the patients consult their family physician or any general practitioner. When special advice is sought the affection is still further advanced, more deeply rooted, and consequently less amenable to treatment.

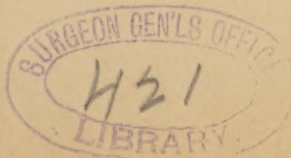
We will probably all agree that if we are ever to be able to successfully treat these diseases—to arrest or cure them—it will have to be by improved methods of treatment applied at the earliest possible moment; as near to the beginning of organic changes as possible, or before a functional disease has become fixed. The latter condition I hold to be a *sine qua non*, and improved therapy will avail but little if it can be applied only to well-developed disease.

Consequently, gentlemen, I have long held it to be a duty of specialists to endeavor by every means in their power to furnish to their *confrères* in general practice the elements for the earliest possible diagnosis of the diseases to the study of which they have devoted all their energies. Nine years ago I read two papers on this subject, one relating to organic nervous diseases,² the other to epilepsy;³ and I have reason to believe that they produced some fruit. The essay which I have the honor to read this evening embodies

¹ Delivered before the Providence Medical Association, December 1, 1890.

² New York Medical Record, February 26, 1881.

³ New York Medical Record, August 6 and 13, 1881. Both in Opera Minora, pp. 457 and 540.



in a briefer form the subjects of those earlier essays, differently arranged, and with some new points and suggestions.

I beg that you will excuse the positiveness of the statements I shall make. All are the result of experience, some of my own mistakes, and every one has been well considered. Besides, the necessity of condensation of so much important matter, renders a terse and rather imperative form unavoidable. In concluding these prefatory remarks, allow me to say that I wish that our masters in other special departments of medicine would give us the results of their experience in a similar way and for the same purpose, that is, to furnish to the general practitioner the means, clearly and logically arranged, of making a correct diagnosis of serious chronic diseases at the earliest possible moment.

Before entering upon the special part of my paper, I wish to make a few remarks upon *diagnosis*, as regards its degree of certainty and its kind or form logically considered. I shall assume that the attempt at diagnosis is preceded by a thorough and exact examination of the patient as well as by the recording of a complete history of the case.

First, we have an ordinary *positive diagnosis*. Most, if not all the cardinal symptoms of a chronic organic nervous disease are present and properly grouped. The case is one of spinal sclerosis, of parietic dementia, of cerebral tumor, or of actual cerebral hæmorrhage or local softening, etc. In such a case it rests with the physician's conscience, his knowledge of his own capacity, whether he shall treat the case himself and be able to do all that can be done to arrest the disease or prevent new lesions.

Second, there is a *diagnosis of probability*. Some of the capital symptoms are present, though perhaps not marked; by exclusion you reject the diagnosis of various diseases presenting similar or analogous symptoms. To your mind it is probable (in varying degrees of probability) that one of the lesions above mentioned is present at a tolerably early stage of its development. In such a case is it right to wait, to allow new symptoms to develop before instituting a course of treatment? Have you the technical acquirements necessary to determine the condition, static or dynamic, of all the patient's nervous organs? In other words, are you able to search for and recognize *every* symptom of the affection you suspect? Here is where the experience of specialists, freely laid before you, may be of use. In

the present state of therapeutics it may be that you can do as much for your patient as a specialist could; but are you on safe ground, are you justified in warning your patient of the probably rebellious course of his ailment, and in obliging him to make the sacrifices necessary to a thorough treatment? In such cases consultations are, I believe, desirable, and equally satisfactory to the patient and to the practitioner. In many such cases the accumulated experience of the specialist will readily clear up what uncertainty may exist. Occult symptoms may be discovered, confirming the diagnosis; or seemingly positive symptoms may be shown to have no serious meaning. I could quote many cases confirmatory of the utility of a consultation under such conditions.

Third, there is, not rarely, a degree of diagnosis which I term a *diagnosis of possibility*. The anomalous course and grouping of symptoms, each one not serious in itself; the absence of elements for the diagnosis of other diseases; the inutility of prescriptions based on less serious lines of thought; all these lead you at once or gradually to suspect that you are face to face with the initial stage of one of the diseases which I shall consider analytically further on. In such a case it is, I believe, an imperative duty to have a consultation. The specialist may be able to dispel your fears, or to advance the diagnosis to the second degrees, yes, in some instances, to the first.

It is for the perfection of diagnosis at and from this stage that I now plead, for the reason already given, that, if we are ever to be successful in curing sclerosis, inflammation of the neuroglia, decay of nervous elements, primary or secondary to arterial changes, neoplasm, etc., it will have to be by energetic treatment, including hygiene, applied at this stage. I need not tell you how dismal and discouraging is the prognosis of the above lesions at the present time; usually, making a correct diagnosis (of the second or first degree), is equivalent to pronouncing the victim's death-warrant.

Besides these three *degrees* of diagnosis, we should bear in mind that the complete study of a given case of nervous disease requires several *kinds* of diagnosis, to be reached by different modes of reasoning. In organic diseases we should clearly establish in our own minds three separate diagnoses:

(1) The diagnosis of the *symptom or symptom-group*. This is arrived at by the history of the case and by a careful examination for the determination of the alter-

ations presented by the patient's functions and external (or accessible) tissues. Physiology is our chief guide here.

(2) The diagnosis of the location or seat of the lesion. This is arrived at by a purely anatomical and physiological reasoning. We trace the symptom (that is, disordered function), to its source or anatomical substratum. I need hardly remind you of the remarkable advances made in this form of diagnosis (so-called localization diagnosis) in the last twenty years. Many small lesions can now be located in the brain and spinal cord with almost geometric exactness.

(3) The diagnosis of the *nature of the lesion*. This is often difficult, and is made by applying deductively the accumulated results of experience as embodied by pathological anatomy and statistics. In nervous disease we cannot hope for a direct demonstration of the nature of the lesion, as we have it in pulmonary phthisis by the presence of bacilli, or in Bright's disease by the discovery of albumen and casts. In nervous diseases the third diagnosis is one purely of inferential or deductive reasoning.

In functional nervous affections, we are limited to two diagnoses :

(1) The diagnosis of the *symptom or symptom-group* made in the same manner as for organic diseases.

(2) The diagnosis of the patient's *general condition and predispositions (heredity)*.

The latter is often made impossible by the silence or deliberate deception of the patient and her relatives. The former is to be obtained by a thorough personal examination: including a study of the state of the blood, of the urine, of the condition of the heart and the arterial tension, the state of digestion and assimilation, and by an examination of those organs which we know are capable of being the starting point of exhausting irritations; for example, the eyes, ears, and sexual organs more especially.

There is, we now believe, no true lesion (that is, gross organic change), in the nervous organs in these cases, yet there must be some delicate alterations underlying the perturbation of functions. In a good many cases of epilepsy, however, a very thorough search may reveal a lesion. What I have just said of forms of diagnosis, as to kind and degree, implies the giving of much time and patient care to the investigation of each case. But I am convinced that you will never have occasion to regret having studied your nervous cases in this way.

Here allow me to say that long before I became a specialist (having been a general practitioner fourteen years before becoming one) I had a very definite contemptuous idea of consultations as ordinarily managed. Some years ago, in all departments of medicine, the advice of a specialist or consulting physician was sought rather to confirm the serious or wholly unfavorable diagnosis of the family physician, and to enable the family to "feel that everything had been done." Consultations *pro forma* were the rule then and they are not unknown now. Is this course in accord with the highest conception of a physician's duty, in that he is bound to do everything in his power for his patients' welfare? If the diagnosis has been correct, and the inevitable fatal issue is approaching, where is the utility of the consultation, and how are we justified in entailing the additional expense upon our clients? If our diagnosis has been wrong from the start, is it not usually too late for the consultation to result in any good for the patient? I leave out, of course, those mortifying cases, of which we all have had our share, when we had believed, and so informed the patient or his relatives, that he had a serious or fatal disease when such was not the case. Here, of course, the consultation serves to dispel the dense cloud resting on a family, and the new diagnosis and plan of treatment are followed by relief or cure: but what are the feelings of the unfortunate physician who had allowed himself to be so positive? In this connection I need hardly remind you of such instances as hysterical imitation of organic disease, emotional paralysis, cardiac murmurs, etc.

At the present time, consultations are being more and more held for what I take to be their legitimate logical purpose, namely, to enable the practitioner to recognize serious disease of unusual occurrence at the earliest possible moment, and to determine the proper line of treatment.

I shall speak only of a few nervous diseases, but I would like to impress you with my own belief that the above remarks apply with equal force to serious diseases of other apparatuses; to medical and surgical conditions. How can we count the victims of the *late diagnosis* of Phthisis, of Bright's Disease, of Perityphlitis, Intestinal Obstruction, Pernicious Malarial Fevers, Glaucoma, Cirrhosis of the Liver, Ulcer of the Stomach, etc., whose lives might have been saved or prolonged, by an early positive diagnosis, or by a strong diagnosis of possibility?

Excuse me if I again state that one of the chief ways in which we may hope to improve our therapy and prognosis will be by the recognition of disease, from the presence of a few carefully ascertained and reliable symptoms, at the earliest possible moment.

However, I do not wish to be understood as claiming that such an early diagnosis can be arrived at only through consultations. The purpose of this, and of my former papers, is to enable the general practitioner to make early diagnoses by becoming possessed of the knowledge and resources of the specialist. Of course, he cannot communicate his maturity of judgment nor all his knowledge of exceptional cases and conditions which sometimes modify an apparently clear case, or throw light on an obscure one, but he can and should diffuse a knowledge of the elements and logic of diagnosis, with far more detail than can be put in textbooks or treatises.

I. POSTERIOR SPINAL SCLEROSIS (TABES).

In spite of the enormous mass of literature relative to this disease which has been distributed to our profession in the last twenty years, this disease is yet, I regret to be obliged to say, often — very often — unrecognized in its first stage, the neuralgic or pre-ataxic stage. Patient after patient comes to the specialist with the same story, namely, that he has been told he had rheumatism or neuralgia, and has been treated accordingly.

Leaving out non-typical cases of tabes, which are highly interesting and often really difficult of diagnosis, there ought not to be any uncertainty attending the very early recognition of sclerotic changes in the posterior root-zones of the spinal cord. The two difficulties in the way of a correct positive diagnosis are (1) the lax and unscientific determination of the symptoms by the physician, and (2) his feeble faith in the fatal significance of the few symptoms presented by the patient. To many a practitioner it seems the height of presumption for another to say that a man who complains of occasional sharp pains in the legs, whose patellar reflex is low or lost, and who has had temporary diplopia, is doomed to be ataxic and then bedridden in the course of a few years. Yet, gentlemen, it is true, ninety-nine times out of a hundred — as true as that a given comet shall return to our limit of vision at a given time.

Allow me treat of the first source of error in some detail, as it is fundamental. It is a result partly of

the faulty clinical teachings in our medical schools, where the science of semeiology is not treated as it deserves: for on it hangs all diagnosis; and partly to hurry and loose habits of questioning of physicians. The patient's statement that he has "neuralgic" or "rheumatic" pains in his legs is allowed to stand as a finality. No cross-questioning is applied to elicit a recollection of past diplopia; no objective examination is made; and two precious symptoms (loss of knee-jerk and of pupillary reflex) are not discovered. The patient goes away with a prescription directed to the relief of rheumatism or neuralgia.

I am one of those who believe that if physicians exercised proper care in the questioning and cross-questioning of patients, and made it a rule always to make a physical or objective examination of every patient, posterior spinal sclerosis would be detected by all practitioners at a very early stage of its development, at that period when possibly we may, by improved therapeutics, some day cure this dread disease.

What are the cardinal symptoms which may serve to justify a positive diagnosis of this disease in the first stage (barring non-typical cases)?

First, and chiefly, the peculiar pains which almost always precede all other symptoms, and may be almost the only symptom for many years.⁴ But to fully appreciate the significance of these pains, and to be confident enough to base a diagnosis on them, it is necessary to study the symptom carefully and minutely. The adult patient⁵ who complains of sharp pains in his legs should be at once *suspected* of having tabes; and most closely questioned; first, by being asked to fully describe the pains in his own way; then, if necessary, to ask a few direct questions tending to bring out sharply the peculiarities of these pains.

The pains of tabes are pathognomonic, if the observer obtains a clear concept of their characteristics, which are:⁶

(a) Irregularity of distribution. The pains appear almost always first in the lower limbs; they strike in the heel, calf, thigh, toes, instep, anywhere and everywhere. So true is this, that, after a few months, many patients will be unable to say where they have not had pains below the groins. In some

⁴I have made an autopsy in a case, which for twenty-nine years presented only two symptoms, namely, fulgurating pains and fixed dilatation of one pupil.

⁵It should be remembered that posterior spinal sclerosis never occurs under twenty years of age, and rarely under thirty.

⁶I arrange these characteristics in no logical order; all are of nearly equal importance.

cases the pains for the first few months affect perhaps half a dozen localities, but always in both legs. A secondary characteristic of this group is that pains never occur simultaneously in the two legs, though the succession may be very rapid. In double sciatica the pain is constantly present along the two sciatic nerves.

(b) Location of the pains. Usually cutaneous, appearing in rounded or ovoid spots or areas; occasionally in streaks of short length; sometimes radiating in a star-shaped way from a given point. Never do the pains extend along a nerve trunk and its branches as in true neuralgia. Occasionally the pains are deeper, in muscles, bones or joints, with somewhat different characters from cutaneous pains.

(c) Nature of the pains. This is perhaps their most important characteristic. The pains are almost always sharp, lancinating or piercing, rarely (in joints and muscles) tearing or crushing. A most valuable criterion is that these sharp pains are repeated in one spot or area every few seconds for hours or days (occasionally for weeks). The patient, if properly interrogated, without leading questions, will say that the sharp pains *strike* the place for so long a time, minutes or hours. This is a peculiarity presented by no other pain that I know.

(d) Hyperæsthesia of the spots or areas which are the seat of pain. The affected places present these peculiarities; a slight contact with the finger, clothing, or even a sheet in bed causes suffering, whereas (usually) a firm pressure does not prove painful, or may relieve.

Let me here point to a contrast between fulgurating and truly neuralgic pains. In the former the tenderness is met with exclusively at the seat of pain; in the latter there are tender points where the nerve-trunk becomes superficial or escapes from an aponeurosis or foramen (*points douloureux* of Valleix). The tenderness of neuralgia is confined to the nerve-trunk or large branches, whereas that of tabetic pains is limited to the skin where the darting pains recur.

(e) The degree of pain is of no special value for diagnosis, though I may remark that the suffering in tabes is very much greater than in sciatica.

With these characteristics well-established in a given patient, I believe that we have in hand irrefragable evidence of beginning posterior spinal sclerosis. Yet there are imitations of the pains, to which I think I was the first to call attention. First, some gouty

persons have sudden sharp pains here and there, which they compare to the stab of a needle, in legs, arms, trunk, or even scalp. This extreme extension alone serves for differential diagnosis; but more important is the fact that the pains occur only once or twice in one spot, and the spot is never hyperæsthetic. Second, in dementia paralytica we occasionally hear complaints of sharp, pricking pains in various parts of the body, much like those of gout, that is, simple and not severe or large. In appreciating the value of such pains in dementia paralytica, it must not be forgotten that in a small proportion of cases the posterior columns are more or less diseased. Third, I should add that I have twice received excellent descriptions of fulgurating pains, "terrible" in degree, from victims of the morphine habit. These men said that the pains occurred everywhere, which is almost unknown in early tabes; and their knee-jerks were too strong. I suspected that these patients, having read about or been told of fulgurating pains, had described their vague pains accordingly, to justify the continued use of morphia.

Barring these three imitations, I repeat that the pains I have endeavored to describe, are, when exactly determined, of pathognomonic value.⁷ But other symptoms are seldom wanting, even in this early stage.

Second. Diminution or loss of the patellar tendon reflex. You all know about this important negative symptom, and how to obtain it. Perhaps all of you may not be aware that where the jerk is absent to ordinary tests, it may still be demonstrated to a feeble degree by diverting the patient's attention by making him count or read aloud, or better, by making him grasp an object strongly while you tap the tendon. If the knee-jerk is absent, you obtain quite a corroborative sign. But let me warn you that this negative symptom is not in itself (and the same may be said of ataxia which appears in the second stage of tabes) of absolute value. This is because the knee-jerk is absent in a variety of affections and conditions, such as old age, diabetes, multiple neuritis, diphtheritic paralysis, lead paralysis, poliomyelitis, etc. It is only a corroborative symptom.

Third. Pupillary immobility or spasm. This is, perhaps, next to the fulgurating pains, the most valuable symptom of beginning tabes; yet, after all, it also ranks only as a corroborative sign. Perhaps it may

⁷ In my experience, after considering all variations in the mode of onset of tabes, and studying all my cases of "abnormal tabes," I hold to the opinion that these pains fall least often of any single symptom.

not be out of the way for me to describe the symptom, which, by the way, must be sought for, as it escapes the patient's observation. The pupils, variable in size, usually contracted, often unequal, present this peculiarity, namely, that, while they contract under the effort at accommodation (looking at your finger at less than eight inches), they neither dilate nor contract under the influence of the strongest contrasting light and shade (tested in a dark room with a mirror, or at a brightly lighted window). This condition of the pupil is often designated as Argyll-Robertson pupil, from the Scotch physician who first described it. I regret to say that its value falls below that of fulgurating pains, as it is also present in some cases of cerebral syphilis and dementia paralytica (and after fifty years of age the "pupillary reflex" is sluggish or almost lost).

Fourth. Transitory or permanent diplopia, either from paresis of an internus or of an externus rectus muscle. This may be present under your observation; but usually it has to be elicited by questioning, having occurred years or months previously. A ready means of determining which rectus muscle was paralyzed, is by asking the patient whether his diplopia was for objects within two feet of him or for them farther away. An adult who has or has had acute strabismus or diplopia should at once be *suspected* of posterior spinal sclerosis or of intra-cranial syphilis, and a careful inquiry made along these two lines. Of course, there are so-called rheumatic paralyses of ocular muscles, and this diagnosis is arrived at by exclusion.

As I am not speaking of refined or "fancy" diagnosis I need go no farther into the semeiology of the first stage of tabes. There are cases in which atrophy of the optic nerves, with fulgurating pains; of mono-arthritis, with fulgurating pains; of vesical paralysis with fulgurating pains; of gastric crises, with fulgurating pains (always in the vast majority of cases, I beg you to remember, *with* fulgurating pains) characterize the onset of the disease. Then there are non-typical or abnormal cases of tabes in which paresis or ataxia or vesical paralysis or gastric and other crises may precede pains; but these are phenomenal cases, each one worthy of record.

(1) In my opinion the determination of the existence of fulgurating pains with one or more of the four symptoms I have referred to in detail, occurring in a subject over twenty years of age, not only justifies but renders imperative the diagnosis of posterior spinal sclerosis. Any other diagnosis should be held as be-

traying ignorance or want of scientific courage. Excuse the force of this remark, but my memory is so filled with recollections of neglected and maltreated patients that I must cry out loudly in behalf of an early diagnosis of tabes.

(2) The existence of fulgurating pains alone, in a subject over twenty years of age, warrants a diagnosis of probability, and justifies the ordering of a special treatment.

(3) The occurrence of transitory or permanent diplopia (strabismus), especially if the subject be over thirty years of age, should at once arouse a suspicion of the beginning of tabes, and other symptoms should always be sought for diligently.

(4) The existence of Argyll-Robertson pupils alone should lead the physician to anticipate posterior spinal sclerosis or dementia paralytica.

(5) The absence of knee-jerk is not in itself of specific value; but it is abnormal, and should cause a careful search to be made for other symptoms.

II. DEMENTIA PARALYTICA.

THIS, the passive form of general paresis, so-called, is often seen by the general practitioner in its earlier stages, and the gravity of the symptoms is almost never appreciated. Even by neurologists the diagnosis of nervous prostration or cerebral fatigue is often made and a delusive prognosis given. Rest and change are advised, when an active medication and seclusion from excitement should be prescribed.

The symptoms by which an early diagnosis can be made are as follows, in order of importance:

(1) A change in the patient's moral character; ethical changes. Ethical development is the last and highest phase of action or function of the cerebrum in mammals, and more strikingly in man; it is the least instinctive or organic function, a sort of delicate efflorescence; and, consequently, it is not surprising that it should be the first to retrograde when the cerebrum is undergoing widespread degeneration of slight degree. The alteration, allow me to repeat, is a positive *change*, not an accentuation to a morbid degree of the patient's previous faults of character, as is observed in various forms of insanity. Diminished regard for decorum, slovenly habits in dress and at table, slight deviations from truthfulness, an inclination to, or relish for ribald or obscene jokes, actual indecency in language and acts, indulgence in stimulants, lascivious familiarities and visits to houses of prostitution, etc., in a man who

previously never lapsed in such matters, should always cause the greatest concern and lead to a suspicion of beginning diffused encephalitis.

Irritability or abnormal anger might be included in this list, but this increased reaction to external stimuli is a symptom more characteristic of cerebral neurasthenia, hysteria, etc., and seldom means a change in character. Indeed, in my experience, good nature and abnormal pliability are more frequent than irritability in dementia. The same remarks apply to the abnormal emotions shown by victims of this disease: they laugh or cry "hysterically" on the slightest provocation. This state, however, only means that we have an abnormally sensitive brain and diminished self-restraint before us—conditions fully as frequent in simple cerebral neurasthenia as in dementia paralytica.

Not rarely these symptoms, though appearing very early, are not known to the physician because the patient cannot tell of them, and his relatives are ashamed to reveal them. They must be sought for; consequently, although these ethical symptoms are of great importance, they cannot be designated as striking or as leading symptoms, except to the family physician, who, of course, has peculiar opportunities for noting these changes, even before the family is alarmed.⁸

Often it is believed by laymen and physicians that the alcoholic and sexual irregularities of the patient are causes of subsequent symptoms, but this is, most authorities agree, an erroneous and dangerous view. Doubtless minute changes in the brain precede the ethical degradation.

(2) Mental dulness and inaccuracy. The patient often complains of these himself; he is becoming "lazy," mental exertion is onerous; he feels dull and even drowsy during business hours; he is conscious of doing everything slowly and laboriously. He notes mistakes in his calculations, failures to keep appointment, and other evidences of failing memory and impaired power of attention. Many cases—those in which a slight degree of exaltation appears early—the patients are unaware of these faults and energetically deny them. Yet it is astonishing how long professional or old habitual acts continue to be performed with tolerable exactness, even after many symptoms have appeared. Such mental operations as have by

⁸ I have sometimes first heard of these symptoms some time after the consultation, from associates and friends of the patient; the family having carefully concealed them.

long practice become almost automatic or semi-instinctive resist the disease remarkably.⁹

(3) Motor disturbances. These are often the first which are complained of by the patient. Difficulty and slowness of articulation; awkwardness of the fingers for delicate movements; impaired handwriting; visible tremor; a general loss of muscular force are often perfectly evident to the patient in those cases where exaltation has not set in. I have notes of several cases in which the patient came to me spontaneously for difficult articulation (this appearing to him the only symptom).

In very rare cases impaired articulation with slight tremor of the hands, are really the first symptoms. I have notes of one such case, in which I made the diagnosis at that stage; the patient dying in Germany several years later with all the symptoms present.

As tremor underlies the faulty speech (or dysarthria) and the awkwardness, allow me to speak of it at some length. Different cases present tremors which I am in the habit of classifying as coarse and fine. Sometimes it requires the closest scrutiny to detect them, or they may be almost choreic in form. Always these tremors appear only on exertion; they are not, strictly speaking, fibrillary contractions, nor are they at all like the rhythmical or quasi-rhythmical movements of paralysis agitans, senile or alcoholic trembling. Consequently you will have to seek for this symptom by bidding the patient to do certain things. Sitting quietly he appears free from tremors; bid him frown, and the muscles of the upper part of the face show tremulous action; bid him show his gums, the tremor appears in all the lower facial muscles; bid him protrude the tongue, and that organ appears filled with fine tremulous contractions, or is agitated as a whole by coarser tremors; make the patient hold out his hands, and various degrees of non-rhythmical tremor are apparent to your eye or to your fingers (if you hold his fingers within yours). Tell him to speak, and the aerial waves are broken by the irregular muscular contraction, producing various forms of faulty articulation. Emotion, which in almost all persons is accompanied by unconscious muscular movements in various parts, also brings out the tremors. At once when the patient begins to tell you his ailments or

⁹ I have known of physicians who attended to ordinary practice fairly well (but were suspected of drinking by their clients) for a long time after the diagnosis was clear; and two years ago I made a diagnosis of dementia paralytica in a popular actor who "starred" about the country with diminishing success for several months afterward.

replies to your questions, the faulty speech reveals the tremors, and to the experienced ear foretells the man's doom.

For, gentlemen, if there is a pathognomonic symptom in all semeiology, it is the peculiar faulty speech of the early stage of non-delirious dementia paralytica. It is as valuable for the diagnosis of this disease as fulgurating pains are for that of tabes. Yet a similar speech (or at least one that seems like it), is met with once in a while in a generally tremulous patient who has not cortical degeneration. So rare is this that I have record of but one example; so that the value of the symptom remains very great.

The fundamental character of the speech in paresis (to use the ordinary short name for the disease) is non-rhythmic vibration. This leads to a jerky irregular utterance, and a still further degree of muscular tremor (or as some wrongly say of incoördination), causes the hardly perceptible sounding or the omission of syllables in long words. It is a mistake, in my opinion, to consider this omission of syllables as the characteristic fault. It is not present at the early period when I would wish my *confrères* to diagnosticate the disease. It is almost impossible to describe by words the characteristics of speech so altered. Last spring I made experiments with the phonograph, hoping to obtain cylinders which might be used to demonstrate to students the faulty speech of this disease, and others, but found that the instrument was not yet delicate enough for that purpose. It requires a good speaker to make a useful record on the machines now in market.

Allow me to repeat that the speech of paresis is tremulous, jerky yet often slow; some syllables are spoiled and some are omitted from long words. No other disease causes this.

(4) Pupillary symptoms. Here we see an indication of the close relationship of posterior spinal sclerosis and dementia paralytica. In point of fact one disease is sometimes complicated by the other and *vice versa*. The pupillary symptoms to be observed are:

Contraction, with loss of passive dilation in the dark. The pupils are often so small as to deserve the appellation of pin-head pupils: shading the eyes or placing the patient in a dark room causes no expansion. They are in a state of spasm as German writers call it.

Inequality is very frequent; it may be difficult to detect, but few cases in my experience have not presented this peculiarity.

In some rare cases (probably truly of syphilitic

origin), the pupils are motionless and unequal but wide. Thus we may say that in dementia paralytica and in posterior spinal sclerosis we meet with unequal, Argyll-Robertson pupils; much more often small in the former disease. Atrophy of the optic nerves is rare, though traces of neuritis are often found.

(5) The patient's manner is very suggestive, quite early in the disease. The patient seems confused in taking a chair, or trying to leave the room;¹⁰ he looks at various objects in your office regardless of the fact that you are questioning him; he looks from his friends to you in a stupid, helpless manner; and tolerates that they answer for him. Frequently he interrupts his friends to deny the symptoms they relate. In showing his tongue he opens his mouth enormously, and makes an extensive absurd grimace. When once seen, it is a behavior never to be forgotten.

As a part of this confusion some patients appropriate objects which do not belong to them, under the idea that they do. This leads to arrests for shop-lifting, etc.

The psychological mechanism of these symptoms is that the patient's perceptions are fleeting and imperfect; his will power diminished; his power of attention impaired; he labors under a sense of unreality and uncertainty.

(6) Varying degrees of exaltation, or morbid optimism. With slow, tremulous, broken speech the unfortunate patient will tell you that he "feels first-rate," "was never better in my life," etc., in shocking contrast to the obvious mental and physical decay. In some cases pretty early, in others later this exaltation grows to extraordinary proportions; the patient claims herculean strength, unmeasured wealth, limitless sexual power (when impotent), supernatural powers, etc. This constitutes the "exalted delirium" with which asylum physicians are best acquainted. But I beg of you not to consider this as a pathognomonic symptom, as some writers would have you believe. Many cases of dementia paralytica never get beyond "feeling very well," some others are neither exalted nor depressed, and a small number have depressing hypochondriacal delusions. In pleading for the early diagnosis of this disease I refer to cases in which exaltation and maniacal excitement, as well as epileptiform and apoplectiform attacks have not yet appeared.

¹⁰ Two years ago I saw a perfectly lucid patient presenting tremors, imperfect speech, unequal pupils, conscious failure of memory, etc. On taking leave of him he put the fee, which his wife had handed him to give me, into his own pocket, and at the door shook hands with her. The poor fellow, who has since died, was instantly aware of these errors and laughed heartily at them.

(7) The reflexes are as a rule greatly exaggerated. Extremely strong knee-jerk, sometimes ankle-clonus, and nearly always wrist-jerk can be demonstrated. In cerebral syphilis and some cases of cerebral neurasthenia we meet with equally high reflexes. The explanation is to my mind simple. I still believe that knee-jerks, etc., are reflex spinal acts (though by far the most rapid of all reflex actions). The healthy cerebral cortex constantly exerts a restraining or inhibitory effect or influence upon the spinal centres. If the cerebral cortex be diseased, by internal inflammatory changes as in paresis, by wide-spread arteritis as in syphilis, or by simple (?) functional exhaustion, this inhibitory influence is reduced or almost lost; hence the increased reflexes,¹¹ and the unrestrained emotional manifestations.

These are the symptoms from which an early diagnosis of dementia paralytica can be made by the practitioner; nay, should be made by him.

(1) A positive diagnosis can be made, I believe, from the speech alone, but perhaps it is too much to ask the general practitioner to risk so much on one symptom. Impaired speech with unequal motionless pupils, high reflexes, and slight mental symptoms should, however, oblige the physician to make a diagnosis, and remove the patient from business.

(2) Fixed, small or unequal pupils with changes in character, increased reflexes, and confusion in manner, should lead to a suspicion of dementia paralytica. Even the small fixed pupils alone should, I think, excite suspicion, and lead to careful observation of the patient.

(3) Mental slowness and inaccuracy, with any one of the other symptoms referred to should cause a strong suspicion of incipient "paresis." The same is true of inexplicable changes in the moral character of a subject above twenty years of age.

(4) Dementia paralytica is, I might add, much more frequent among women than is generally held by authorities. They can more easily cover up signs of mental failure, and they seldom exhibit exaltation. Guided by the points I have given as of great diagnostic value, you will be able to recognize a good many female cases.

(5) A general character of great value is the gradual slow onset of symptoms. When an adult rapidly be-

¹¹ In a small minority of cases the knee-jerk is absent or much reduced and the patient describes sharp pains in various parts of the body. These are cases in which the posterior columns and roots of the cord are affected by sclerosis; it is yet a question whether this is a commencing or whether it is an extension downward of the cerebral changes.

comes demented (foolish in manner, inattentive to his person, even to the point of not controlling his evacuations), has unequal pupils, and large quasi-choreic ataxic tremors with early convulsive seizures, it is possible that the case is one of cerebral syphilis, which may be cured by heroic treatment.

(6) You should not be discouraged in your diagnosis by an apparent return to health after a few months, because extraordinary remissions, lasting several months, occur in the course of dementia paralytica, yet, even in these remissions, a critical examination almost always reveals traces of the physical symptoms.

III. CEREBRAL TUMOR.

I had intended saying a good deal about the early diagnosis of cerebral tumor, but the subject is so large that there would not be time for its careful consideration this evening. Let me just enumerate the following principal symptoms of encephalic tumor, namely:

Headache, local or diffused.

Paralysis of various distribution, and almost invariably progressive in development.

Convulsions, usually having the same distribution as the paralysis; occasionally general spasm. Either of these symptoms may precede the other, and no special value attaches to the order of precedence.

Opisthotonus.

Vomiting, usually without nausea.

Anæsthesia, very rare; usually of same distribution as the motor symptoms.

Mental dulness, stupor, and even transitory coma.

Aphasia, amnesic, sensory or motor.

Hemianopsia, dark half-fields on side opposite the tumor, and on same side as the motor and sensory symptoms. If the optic nerves are normal, the pupillary reaction is completely preserved.

Choked disk or neuro-retinitis, with hæmorrhages in the retina; succeeded by

Atrophy of the optic nerves; but this symptom may appear primarily.

Paralysis of one or more cranial nerves independently of body-paralysis.

Increased temperature of scalp over supposed site of tumor.

Localized tenderness to percussion over supposed site of tumor. Both these symptoms are unusual and of uncertain value.

Slow pulse occurs when the intra-cranial pressure is

much increased, and often is co-occurent with deep stupor lasting hours or days. Repeated attacks of stupor with slow pulse during the course of a case of encephalic tumor, are very strongly indicative of distension of the ventricles by fluid.

Unfortunately none of these symptoms is pathognomonic; not one has nearly the value that fulgurating pains have in tabes, or defective speech in dementia paralytica.

At one time it was hoped that choked disk would prove of greater value than any other single symptom, but we have known for ten years that many cases even of enormous tumors do not present choked disk, and that, on the other hand, this ocular lesion occurs in persons who have no intra-cranial disease. Indeed, in my experience as regards tumors of the hemispheres the rule is, that the optic nerves are normal. When the tumor is situated in any other part of the encephalon, choked disk seldom fails to appear; often very early.

A diagnosis must consequently be made by studying the grouping and the mode of progress of the symptoms. The latter I believe to be most important. For instance, the co-existence of recurring localized spasm (Jacksonian spasm) with a progressively increasing paralysis of the same part as that which is convulsed (let us say one arm or one leg for example), may sometimes justify the diagnosis of cerebral tumor, even if all the other symptoms are absent.

Local or Jacksonian spasms, occurring in a previously healthy adult patient, should always excite suspicion of brain tumor. The same is true of choked disk alone (when Bright's disease can be excluded).

With reference to the value of localized spasm it should be added that there are a few cases on record which would seem to indicate that they may occur in hysteria—very rarely of course. Besides, if the paralysis following the spasm (of one forearm let us say) is only slight or transient, we must remember that a superficial or cortical lesion, not a tumor, may cause the symptoms. I have reported such a case in which a patch of localized meningitis over the facial centre was diagnosticated during life and verified post mortem.¹² I should add that the diagnosis of cerebral tumor may now be made so early (that is, when the tumor is very small) that immediate surgical interference may be of no service. In a case of mine operated by Dr. R. F. Weir, the tumor was deeply seated in the

¹² Journal of Nervous and Mental Diseases, vol. xiv, June, 1887. [Case I.]

white substance, and was not larger than a small almond. It seems a miracle that we detected (by palpation) this small sub-cortical sarcomatous mass.¹³ Recently Dr. Joseph D. Bryant operated on a case in which I had (in conjunction with Drs. Fuller and Wolff, of Hartford) diagnosticated a tumor under the motor centre for the left leg. The operation failed to reveal the tumor, though most careful palpation was practised and incisions made through the cortex. A careful autopsy made on the partly hardened hemisphere revealed a gliomatous mass about half an inch in diameter, just about in the anticipated location. The tumor being of about the same consistency as the surrounding white substance, could not be detected by palpation. Again, about a year ago, Dr. Weir operated in a case in which I felt quite sure that there was a tumor. None was found at the operation, and we were naturally much disappointed. A regular autopsy was not allowed, but Dr. Weir removed the whole of the right hemisphere with his fingers, through the large trephine aperture, without finding a tumor. However, judging by my last case, the one operated in Hartford, I feel quite certain that a small sub-cortical glioma or sarcoma did exist in this case, and the coarse post-mortem examination could not have revealed so small a mass. It is highly desirable that in all such cases the brain should not be cut in pieces at the time of the autopsy, but placed in hardening fluid for more careful study later.

The practical conclusion to be drawn from these three cases is, that surgical interference may be attempted too early, that is, when the tumor is too small to be recognized, especially if it be sub-cortical.

IV. VERTEBRAL DISEASE (CARIES, ETC.).

It may seem strange that I should call your attention to conditions which apparently belong to quite a different specialty from neurology; but, gentlemen, the first and the last symptoms of these diseases (spondylitis, caries of the spine, vertebral cancer) are nervous symptoms. The case appears at first as one of rebellious neuralgia or muscular rheumatism, and lastly as one of paraplegia. The early symptoms do not very distinctly point to the vertebræ as the site of disease; and thus usually the cases remain a long time — during the best time for successful treatment — in the hands of the general practitioner; then, later, they are passed on to the neurologist or orthopedist.

¹³ American Journal of the Medical Sciences, July, August and September, 1888.

Many precious months are thus lost. Yet, if the few symptoms present during the first stage of these diseases are rightly appreciated and correctly interpreted, I believe a diagnosis should always be possible long before angular curvature (representing the breaking-down of one or several vertebræ) or tumor appears. The early symptoms of spondylitis or tumor are the same in *kind*, no matter what part of the spine is affected, but their distribution varies according to the location of the lesion up or down in the vertebral column. This distribution is so peculiar as to enable us to know with almost absolute certainty which vertebræ are affected.

The capital symptoms of the first stage of Pott's disease or of vertebral cancer are only two in number, namely: (a) a fixed pain seemingly of a neuralgic character, far away from the spine. (b) Rigidity of certain muscles attached to the spinal column; a reflex protective or conservative spasm. The distant pain is increased by attempts to overcome the muscular spasm, and by jars.

It will be necessary to consider these symptoms as distributed when the disease (caries, tubercle or cancer of the vertebræ) attacks different regions of the spinal column.

(a) The "neuralgic" pains, and spasm.

(1) Disease of the uppermost cervical vertebræ, spondylitis colli, is not rare. The patient complains in the first place, and chiefly of pain in one occipital region, aggravated by motion or jar. On analysis, we find the pain to follow the range of distribution of the greater and lesser occipital nerves; one or both. Occasionally there is also pain in the temple of the same side. Almost invariably this neuralgic pain for which the patient asks relief, is unilateral.

Inspection reveals at quite an early period a slight or decided "wry-neck," a deviation of the head from its proper vertical position. It is a peculiar oblique attitude, dissimilar from that produced by (functional) spasm of one sterno-mastoid. Any attempt to correct this deviation, and indeed any passive movement of the head and neck cause greatly increased pain in the occipital region (not in the spine). Sudden pressure on the top of the head by the physician's hand causes excruciating pain of similar distribution. The spine itself is not tender or deformed. The patient tells you that the jar of a carriage or horse-car, or of a false step causes intense agony. Some patients very early acquire an instinctive habit of supporting or

steadying their heads with their hands, to avoid effects of shock. Further examination shows that the deeper cervical muscles, extensors, flexors and rotators are in a state of constant spasm, more especially on the side of the pain.

Let us see if anatomy helps us in diagnosis. The occipitalis major nerve is mainly a branch of the second cervical nerve; the minor, of the first cervical nerve. They, however, have branches of intercommunication. Most filaments of these nerves are sensory, supplying the scalp of the occiput and parietal regions. Motor fibres from these two cervical nerves supply the small, deep muscles which govern the movements of the skull upon the vertebral column.

Consequently, both the "neuralgia" and the spasm point infallibly to disease in or about the two upper vertebræ. The exact nature of the lesion may be in doubt, but we have by strictly scientific methods located the disease; it is so placed as to irritate the first and second cervical nerves.

(2) Disease of the lower cervical vertebræ is very rare. In such a case the pain would be in the lower part of the neck, or in one arm or hand according to the exact location of the lesion. The spasm would be in the lower cervical muscles and in those of the arm.

(3) The most common location of these lesions is in the dorsal region, between the fifth and the twelfth dorsal vertebræ. Many and many a child is treated for months for "colic" because he complains of a pain in one side of the abdomen. The too frequent neglect of thorough objective examination here leads to the erroneous diagnosis of intercostal or abdominal neuralgia, or of colic; even of "hepatalgia," according to the exact seat of pain. The muscular symptoms are present here also, but not in as striking a shape as in spondylitis colli. They must be sought for by careful examination. This reveals one of several conditions or several combined. The respiratory thoracic or abdominal movements on one side (rarely on both) are hindered, and the muscles appear to palpation hard or rigid. The various movements of the spinal column are not normally free. Turning the head about as if to look for something is done by a turning of the whole body, flexion and extension (latero-flexion more especially) of the spine are checked by pain or directly hindered by rigidity of the erector spinæ muscles. A segment of the dorsal spinal column is rigid during all attempts at movement. The tenderness of the dorsal nerves cannot be demonstrated by

direct testing with finger pressure, but it is strikingly revealed by what I call the heel-jar test. This consists in placing the patient standing in the military position of "attention," on a hard floor. Then tell him to rise on his toes and then suddenly to drop his whole weight on his heels. If there is vertebral disease, decided or excruciating pain is caused by this jar, not in the spine but in the location of the "neuralgia" for which the patient consults you. This heel-jar test is useful in any location of the vertebral disease. The origin of the nerve which is the seat of pain, the range of the muscular rigidity will enable us to localize the lesion to the exact vertebra or vertebræ.

(4) The lumbar vertebræ are sometimes diseased. In such a case the pain would be in the groin and anterior and inner parts of thigh; the spasm in the same parts; especially in the *psosæ* and *iliac* muscles.

(5) Caries of the sacrum gives rise to pains in the perineum, posterior part of thigh; and in the leg and foot. Cramps or spasms would occur in the same parts (seldom present).

The general diagnostic law may be formulated as follows: The seat of neuralgia and of spasm, though occasionally not in corresponding parts,¹⁴ clearly refer to irritation (compression) of one or more spinal nerves on one side. A knowledge of the distribution of spinal nerves enables us to state with great accuracy which vertebræ are diseased.¹⁵

(b) With reference to paralytic symptoms.

Occasionally they appear before actual destruction of bone brings about angular curvature; the spinal cord being compressed by inflammatory or caseous masses originating in pachymeningitis, or by a tumor. When the disease affects the two upper cervical vertebræ, the paralysis may be hemiplegic, face not affected. This is because the caseous masses have formed on one side of the canal and exerted pressure on one side of the spinal cord, where the large crossed pyramidal fasciculi run downward; hence hemiplegia. Below the level of the second vertebræ the masses which compress the cord are formed anteriorly as a rule, and cause pressure almost equally on both sides of the median line; hence paraplegia; of the type "cervical paraplegia,"

¹⁴ The parts which are the seat of neuralgia and spasm do not always correspond in a topographical sense, but they always do in a physiological sense in obedience to Van der Kolk's law, namely, that the sensory branches of a mixed nerve-trunk (or of a plexus) supply the skin of the part which is moved by muscles which receive its motor filaments. This law finds a wide application in neurological diagnosis.

¹⁵ Consult the diagrams and schemas of spinal nerve distribution in several recent text-books on neurology.

where the whole body below the neck is paralyzed, or "common paraplegia," when the lower limbs and a varying extent of the trunk are paralyzed. It is important to determine the uppermost limit-line of the paralysis, as this usually indicates the limit of intra-vertebral lesion.

When caries exists in the mid-dorsal region, vesical paralysis (retention) is, in my experience, a very early symptom; sometimes existing without other paralysis. With disease of the upper cervical vertebræ we also observe paralysis of the small, deep muscles connecting the head with the spine ("loose head").

When the lumbar vertebræ below the second, or the sacrum is the seat of caries (or cancer), a very peculiar paralysis results. As there is no spinal cord below the level of the first lumbar vertebræ, pressure below this point will affect only nerve-bundles: the constituents of the *cauda equina*.

Physiologically, therefore, the resultant paralysis is a peripheral or neural paralysis (precisely the same as when an outside nerve-trunk is injured), characterized by a flaccid atrophic paralysis, with degenerative reactions; co-extensive anæsthesia; absence of all reflexes; relaxation of the sphincter ani and vesical paralysis. The paralysis is nearly all below the knees, as some of the thigh-muscles are supplied by the crural plexus.¹⁶

We can thus — I hope to have made it clear and easily understood — readily make a diagnosis of a vertebral bony lesion or of an intra-spinal tumor at a very early period; months before angular curvature (kyphosis) or external tumor shows itself. In my opinion there is no justification for waiting till kyphosis appears before reaching a diagnosis. The exact seat of the lesion we can, also, by the help of anatomy, always determine with accuracy.

The further diagnosis, namely, that of the nature of the vertebral lesion, is a most interesting, but complicated problem, which I cannot enter upon to-night. The neural irritation or spinal-cord compression, may be due to pachymeningitis, to vertebral caries (spondylitis), to peri-vertebral or intra-vertebral tumors, or to cancer of the bodies of the vertebræ themselves.

Let it suffice, if I have made it clear, that occipital neuralgia, with rigid, painful wry-neck; intercostal or abdominal local pains (neuralgia so-called), one-sided pains along some nerve of the lower extremities; with

¹⁶ If the previous symptoms — neuralgic pains and muscular spasm — be ignored, there is some danger of confounding the case with one of sub-acute or chronic polyomyelitis; though in this disease anæsthesia and vesico-rectal paralysis do not occur.

associated spasm, mean, or at least suggest, vertebral disease of some sort, and call for a careful objective examination, instead of an off-hand prescription, for the symptoms complained of.

V. EPILEPSY.

It is perhaps more important to make an early diagnosis of this protean affection than of any of those previously studied, because there is no doubt but that by early, careful, and long-continued treatment it can be, in a small minority of cases, cured. What this proportion is no one can tell. In my own experience I have records of several cases which have been perfectly free from attacks (from any manifestation of epilepsy), for upward of four years; yet I am hardly prepared to report them as cures. Recently, one of my cases relapsed after an interval of over ten years, but as I have only the patient's bare statement, and she lives far away, there is just a possibility that the attack was hysterical. Still, I do believe that some cases are cured.

Epilepsy is a chronic disease characterized by the recurrence, at irregular intervals, of attacks, (seizures or popularly speaking "spells" or "fits"). These attacks vary extremely in form; some are terrific in their violence, others so slight and transient as to escape the observation of even experienced physicians. The various forms may, however, be classified with sufficient accuracy under the five following types or varieties.

(1) Simple motor epilepsy (epileptiform spasms without loss of consciousness): Jacksonian epilepsy. These spasms are localized in various parts of the body. Sooner or later loss of consciousness follows the spasms, thus demonstrating its relationship with:

(2) Common spasmodic epilepsy (*grand-mal*).

(3) Attacks, with slight momentary tonic spasm, or consisting only (?) of a peculiar momentary sensation in the head. In both these is a short loss of consciousness, though the patient frequently denies it. This is sometimes called epileptic vertigo (a misuse of the word vertigo) or *petit-mal*.

(4) Psychical epilepsy; in which a seemingly volitional co-ordinated action (often complex) constitutes the seizure; or it may appear as a temporary insane condition or psychosis.

(5) Hystero-epilepsy; a hybrid form in which symptoms of epilepsy and of motor hysteria are variously combined.

These types may co-exist in one individual, any two or all of them. Careful inquiry reveals the occurrence of *petit-mal* in many cases of *grand-mal*.

However varied may be the combinations of types of attack, the successive seizures of each type almost exactly resemble each other in a given patient: we say that epileptic attacks are, as it were, stereotyped. Careful attention to this point will help in its diagnosis from hysteria and malingering. For example, if a patient have at different times attacks of *grand-mal*, of *petit-mal*, and of psychic epilepsy, the succeeding seizures of each type will be almost absolute copies of the preceding ones of that type. As we sometimes say of the heart's action, there is a regular irregularity in the symptoms.

Some authorities reject simple motor spasm¹⁷ from the class of epileptic manifestations, but any one who has watched the evolution of a case of symptomatic epilepsy (from a cerebral tumor for example), will be convinced that the loss of consciousness, clonic movements and asphyxial stage, are but the crowning feature of an extension, topographically and in severity, of the spasm which at first was very local, for example, in one hand and forearm; or, in other cases, simple motor seizures alternate with typical attacks of *grand-mal*. The proof of the epileptic nature of psychic seizures is also obtained by our knowledge of their co-occurrence, alternations with, and substitution for common attacks, as well as by the therapeutic fact to be referred to further on.

We must bear in mind that no one symptom of epilepsy is pathognomonic or even has as much value as fulgurating pains have in tabes, or dysarthria in dementia paralytica. It is the co-occurrence and grouping, or the sequence of the symptoms which go to make up an inductive diagnosis, even in what at first sight seem very different conditions. What could be more different, on the surface, than an attack of *grand-mal*, an epileptic vertigo, or a maniacal manifestation of epilepsy?

Next, allow me to speak in detail of the diagnostic value of the individual symptoms of an attack.

(1) Loss of consciousness, is by some held to be a never-failing symptom, but if we admit Jacksonian spasms into the epileptic group, it will be found wanting in all cases of purely motor epilepsy. In some cases of *petit-mal*, those in which a momentary

¹⁷ Of course every spasm is not epileptic; its form, distribution, and especially its evolution and association must be considered.

sense of stoppage of cerebral action, or a peculiar sensation constitutes the attack we cannot feel sure that

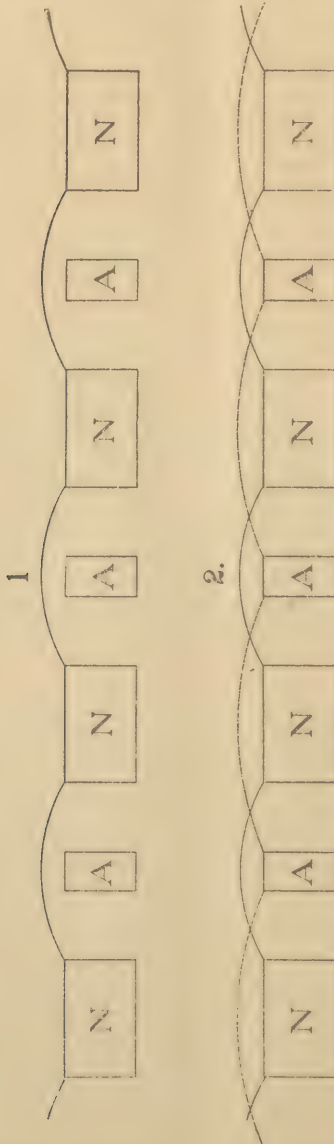


FIG. 1. State of Consciousness in Epilepsy, Coma and Syncope. Absolute intervals in psychic life.

FIG. 2. State of Consciousness in Somnambulism, Hypnotism (of a certain degree), some cases of Psychic Epilepsy and of Insanity; Double Consciousness. The states of abnormal psychic activity are united to one another but separated from normal consciousness by as complete a break as in condition one.

N, Normal conscious state.

A, Attack (blank, or abnormally conscious).

consciousness is lost; we *infer* its momentary suspension in contradiction to the patient's positive as-

section. In some cases intermediate between *grand-* and *petit-mal*, patients sometimes prove by repeating after an attack what was said or done before them, that the unconscious period was much shorter than we supposed. In psychic epilepsy it is not a simple loss of consciousness which obtains, but a peculiar different consciousness which cannot be recalled or reproduced after the attack; in other words, there is amnesia of all that took place in the seizure even if it lasted days, weeks or months. During the attack the patient has a consciousness belonging to the morbid condition of the brain underlying the attack; he *seems* conscious, answers correctly, does what he is bid, performs the ordinary acts of daily life, even to executing business matters and travelling. It is a state of consciousness not unlike that observed in trance, somnambulism and deep hypnotism. It is an unsolved problem whether the abnormal conscious state is recalled by the patient in subsequent paroxysms; in other words, whether we have here "double consciousness," in which the pathological state of the ego is continuous in succeeding attacks, just as the normal consciousness is continuous in the periods between attacks. An absolute barrier, as impassable as that between life and death, which we term amnesia, separates the two states. What a startling problem this double life presents to the student of the human "soul." (See cut.)

I am disposed to classify procursive epilepsy, as newly described by Bourneville, with psychical epilepsy. The running is probably done in obedience to voices heard or visions seen (hallucinations).

After a paroxysm of psychic epilepsy some patients apparently recall a few features of the attack, but I am inclined to think that this is really a process of inferential reasoning rather than a true, direct recollection. In the same way some patients know that they have had an attack of *grand-mal* or of *petit-mal*.

The extreme suddenness with which consciousness is lost in ordinary epilepsy is of considerable value for its differential diagnosis from syncope and hysteria.

(2) Dilatation and immobility of the pupil. This invariably occurs only in *grand-mal*, and forms an intermediate between this and *petit-mal*. It is often present in true *petit-mal*, and is not observed in most cases of psychic epilepsy. It is a symptom generally overlooked by the laity; and occasionally (as we seldom are able to witness attacks), it is desirable to instruct relatives how to detect the symptom to help us in diag-

nosis. In cases where there is doubt, for instance, as to whether a convulsive attack is epileptic or hysteric, the determination of this sign is vital. It should be looked for at the onset of the seizure.

(3) Co-existent with dilatation of the pupil is pallor or an ashen-gray (livid) hue of the skin of the face. Why it occurs in the same cases that show pupillary dilatation is explained by the generally accepted theory that both symptoms are due to the same cause, that is, vaso-motor spasm occurring as a part of the general motor discharge at the onset of the attacks. The motor discharge (from the epileptic centre?) goes out by two channels: (a) through the common motor nerves supplying the striped muscular system generally (except the heart), and (b) through the vaso-motor nerves supplying the unstriped muscular fibres of the vascular system. In the iris it is a question whether the spasm is vascular (my own view, following Rouget) or simply muscular (dilating fibres contracted). Here, again, we have a symptom which, practically, is not of very great utility because it is often overlooked by witnesses: they often persist in saying that the patient's face was red or flushed or normal during the attack. This faulty observation is due to the fact that the observation is not made at the really initial stage of the seizure. It is a fleeting symptom, quickly succeeded in most cases by ordinary color, flushing, or intense turgescence.

(4) The spasm itself. It is most important to obtain as minute a description as possible of the first spasmodic movement of an attack supposed to be epileptic. The questions should tend to elucidate (a) the exact point of departure of the spasm (first movements or "signal-symptom" in organic epilepsy); and (b) the form or nature of the spasm. If you learn, for example, that the spasm always begins in the right fingers, you have a precious guide for your later pathological diagnosis. In the majority of cases the first movement is bilateral and general, often first noticeable in the eyes, neck and throat. It may be momentary, a simple stiffening of the body; congealing, as it were, the patient in the attitude he happens to be in ("statue-like state").

As regards the form of spasm, in typical *grand-mal* careful observation always reveals two modes of muscular movement. One is a sudden rigid contraction (tonic spasm) of the entire muscular system, including the laryngeal and thoracic muscles. This constitutes the whole spasm in many cases of *petit-mal* ("staring-

spells," "statue-like state"). It is momentary in duration, seldom lasting half a minute, contrary to the assertions of lay witnesses. In *grand-mal* there next occurs jerking or intermittent spasms of nearly all parts (clonic spasm), which may last a minute or more in rare cases. Coexistent with this we have congestion or purplish hue of the face, frothing, biting of tongue, emission of contents of viscera; continued unconsciousness. These symptoms of the second stage of a true epileptic attack are due to the asphyxial state produced by the first or tonic spasm. In *petit-mal* we have only the first or tonic spasm. In psychic epilepsy, though there may be complicated muscular movements, we never have spasm: the movements are co-ordinated and apparently voluntary.

It is of much importance to obtain a clear description (assisted by well-directed questions) of the form of spasm observed. In hysteria, for example, the tonic period is often absent, and the jerking convulsions are usually more or less co-ordinated, dramatic and quasi-voluntary. The asphyxial condition is not present, nor of course do we have the vaso-motor spasm (pallor of face or dilatation of the pupils). Malingerers seldom know enough to produce the proper succession of tonic and clonic spasm, though they may produce a terrific convulsion and froth pretty well. In both hysteria and malingering the spasm is much prolonged—far beyond the limit of from one and a half to two minutes rarely observed in epilepsy. Often, also, the spasms are much more violent in the non-epileptic states. As a part of the spasm, the condition of the eyelids is of paramount importance for diagnosis between hysteria and epilepsy. In the latter they are always (?) open, usually staring and fixed by tonic spasm, while the closed, quivering lids of the former condition are significant to the skilled observer as soon as he glances at the convulsed patient. I have been led by experience to attach very great value to these points.

(5) The sensory aura. I have spoken of the value of the "signal-symptom" or initial local spasm, as a help to the diagnosis of organic epilepsy and of the location of the lesion. The aura, so called, is of some importance also, by enabling us to locate quite accurately the seat of primary irritation in the sensory portion of the central nervous system. Just as a tingling sensation in the little finger is characteristic of a blow upon or an irritation of the trunk of the ulnar nerve, so do the sensory auræ of epilepsy point to irri-

tation of some sensory nucleus or path. The sensory aura often coincides in location with the first spasm or "signal-symptom" (for example, in one hand). In most cases, however, the sensation just preceding the loss of consciousness is quasi-visceral; apparently starting from the stomach, the abdomen, or one iliac region. When the aura is persistently placed deep in the so-called ovarian region (which is not at all the region of the ovary) it gives rise to the idea that the epilepsy is caused by ovarian disease. On such a flimsy basis, re-enforced by the fact that attacks are more frequent, or wholly confined, to the menstrual period, a whole theory of ovarian epilepsy was erected a few years ago and many women needlessly mutilated by surgical treatment. I have seen several cases in which ovariectomy, single or double, had been performed without the slightest effect on the disease. To be logical, the stomach, small intestines, sub-sternal parts, limbs, etc., should be excised to cure epilepsy.

The true interpretation is, that a centrally-placed irritation produces a referred sensation in the distribution of sensory nerves arising in or passing through the seat of the lesion. Thus the very common sub-sternal and gastric aura represents, to my mind, a lesion of the floor of the fourth ventricle, and autopsies go to support this view. I have a case of *petit-mal* under treatment in which for several years the aura was hypogastric (or apparently uterine). It gradually ascended to the middle of the abdomen, and is now nearly a gastric aura. This cannot be explained by any theory of extension of the lesion from the uterus to the intestines, and thence to the stomach; but it is very significant of a transfer of the lesion causing attacks, to a different level of the medulla oblongata.

If you will allow me here a therapeutical digression, I will say that the determination of the signal-symptom and of the sensory auræ is of value as a guide to the abortive treatment of attacks on the principle advanced by Brown-Séquard, namely, that an artificial irritation applied to the seat of aura (or signal-symptom) often arrests the attack. For example, in cases where the signal-symptom, or aura, is in the hand or in the foot, the sudden application of a ligature on the wrist or ankle often prevents or aborts the attack. The fact is ancient, but the true theory of its application was first given by Brown-Séquard; a centrifugal irritation inhibits the central discharge. I have been very successful in such cases by having the patient wear a bracelet or anklet of metal or cord,

to be suddenly and severely tightened as soon as the patient has the first sign of the attack. Gastric and sub-sternal aura are best met by an irritation to the fauces, a swallow of table-salt, or an inhalation of spray of carbonic acid. The application of these mechanical means, as well as the inhalation of nitrite of amyl (irrespective of the location of the aura), is limited to the few cases in which some little time intervenes between the occurrence of the premonition and loss of consciousness, and the patient has time to call for help, or to open a bottle and smell it.

(6) The post-spasmodic stupor is of considerable value in estimating the probability that a nervous "attack" is or is not epileptic. In the vast majority of cases of *grand-mal* a deep sleep of variable duration succeeds to the spasm and subsequent coma. In some instances the patient apparently regains consciousness, utters a few words or sentences, then lapses into sleep. On awaking there is no recollection of the remarks made. In other cases only a transient sense of dulness or sleepiness is experienced; and this is true of many cases of *petit-mal*. Very often in *petit-mal* there is not a trace of stupor after the momentary seizure; conversation being continued, or a muscular action kept up right away. There are cases of *grand-mal* in which no sleep occurs, but even in these rare cases a certain sluggishness in action and speech is evident for a time after a seizure. In hysteria, on the other hand, the patient usually is clear-minded and bright even after severe spasms, and always recalls what she may have said between recurring paroxysms.

In some cases of epilepsy and of hysteria, delirium follows the spasm, or *petit-mal*. In some epileptics transient seizures, overlooked by lay observers or careless physicians, are succeeded by delirious, co-ordinated actions seemingly intentional and deliberate; such as wandering off, stealing, committing murder (sometimes on a disliked person, thus apparently proving *intent*), yet, when the patient "comes to himself," hours, days or weeks after, there is complete amnesia of the things done and said. I am inclined to agree with Krafft-Ebing in holding amnesia to be a *sine qua non* for the diagnosis of epilepsy in such cases.

(7) Post-epileptic sensations. Often after *grand-mal*, patients feel sore, either generally, or more especially in one member (if this has been the seat of the chief spasm), as if they had been beaten, to use a common expression. Often also, there is headache of no definite distribution, and also a sense of exhaustion and

languor. These symptoms help us to determine the occurrence of nocturnal seizures in the absence of witnesses. I have known the recurrence of soreness in the ball of one thumb, on a number of mornings, to lead to the diagnosis (verified by witnesses later) of nocturnal *grand-mal*. A case came under my observation two years ago in which, on some five or six occasions, the patient (a bachelor) awoke with one humerus dislocated, suffering intensely from this, and also from general muscular soreness. On one occasion both humeri were dislocated. Strange to say, the diagnosis of nocturnal *grand-mal* had not been made until all but the last dislocation occurred.

Other valuable evidences of nocturnal *grand-mal* are (1) the occurrence of minute specks or points of hæmorrhage under the skin of the face and neck, discovered on rising. This sign has an almost pathognomonic value. (2) Emission of urine in bed, without awakening. This in adults who have not had the habit of "wetting the bed," is strong presumptive evidence of an epileptic attack. In children it is, of course, a much less significant sign.

The occurrence of any of these symptoms (aching limbs and soreness, speck-like hæmorrhages, enuresis), should make us insist on having some one sleep with the patient long enough to obtain an observation, before a positive diagnosis is made.

(8) Is any value to be attached, in the case of a patient who sleeps alone, to a dim recollection of an aura, or of something like an attack having occurred during sleep? Not infrequently our patients report some such dream-like reminiscence. I think that, usually, this is really a dream, that is, the patient dreams he has had an attack, and wakes with some faith in the dream. In my experience such reports have been made by patients who did not present the usual signs of a recent seizure. Unless the case be one of very strong and long signal-symptom or aura, I think that recollection of an attack is impossible; and it is generally recognized that amnesia is peculiarly complete in cases where it is known that nocturnal attacks do occur.

(9) There is still another means, not generally known, of distinguishing between hysterical and epileptic attacks, especially in those hybrid cases (hystero-epilepsy), in which symptoms of both orders are commingled; also of determining the truly epileptic nature of attacks which have occurred without witnesses, or are described by careless and incompetent

ones. I mean the therapeutic test. As far back as 1873, I satisfied myself that an anti-epileptic treatment (bromide treatment) aggravated hysteria, particularly its spasmodic form, and subsequent experience has strengthened my opinion. To state it briefly: a tonic treatment, especially by strychnia in full doses, benefits hysterical cases, while it causes (except in a small number of cases of *petit-mal*) increasing numbers of spasms, and more severe spasms in epilepsy. Contrarily, a bromide treatment reduces the number of, or completely prevents, attacks of a truly epileptic nature, and the same treatment aggravates hysteria. I would not be too dogmatic about the value of this rule, but it is certainly one of much practical value. Especially have I been satisfied with its application in the (not rare) cases of epileptoid spasms of young children between eight and fourteen, which are, especially in boys, much more often than is supposed, hysterical. In such cases we also have, as guides to a correct diagnosis, very prolonged spasms, co-ordinated or dramatic spasms, absence of enuresis and of post-epileptic stupor, and the fact that the eyes are *firmly* shut in the seizures.

It will be said that I have given too much time to the analytical study of the symptoms of epileptic attacks. The disease is so common, and its diagnosis so often difficult, that I think I am justified in detailing the foundations for a correct inductive diagnosis.

Let me add a few remarks of a more general nature.

The errors usually made in the study of epileptic cases are two, namely: (1) Underestimating or not appreciating the meaning of a first attack of *grand-mal*; (2) not recognizing the epileptic nature of slight, simple *petit-mal*.

(1) I have elsewhere treated at some length of the difficulties attending the diagnosis or estimation of a first convulsion in childhood.¹⁸ I have there related several cases in which attacks of *grand-mal* were treated by able physicians as eclamptic or symptomatic; usually of gastric origin. In many cases no proper treatment is directed until quite a number of spasms have occurred, spread out over long but diminishing intervals, and the "epileptic habit" has become established. I will not repeat the arguments¹⁹ I there advanced to show that after the third year of life,

¹⁸ Opera Minora, p. 540, et seq. (New York Medical Record, August 6 and 13, 1881).

¹⁹ Loc. cit., p. 549, et seq.

symptomatic (eclamptic) spasmodic attacks are rare. They do occur occasionally, in very excitable, neurotic subjects after that age, but I repeat, very rarely, until we reach the age when uræmia and syphilis appear as potent factors of epileptoid seizures. In the first two years of life there is great convulsibility, if I may be allowed the word, and such causes as intestinal, preputial (vulvar), dental irritations may give rise to spasms which are precisely like those of epilepsy. During the same period, and a little later in irritable subjects, the onset of acute disease (exanthemata, pneumonia, malarial fever, etc.) may be characterized by an epileptiform seizure. Consequently, if the attacks are very few in number, and *always* accompanied by a decided exciting cause, we may call the attacks eclamptic, and hope that there will be no succeeding ones. But after the third year, and during the first few weeks of life, an epileptiform attack, is, in my experience, the beginning of the long and fatal chain of attacks which we call epilepsy. After a second seizure, in my opinion, especially if the attacks were not accompanied by very strong evidences of peripheral irritation sufficient to cause a spasm, a cautious bromide treatment should be begun and kept up for several years.

In adolescents and adults the diagnosis of a first or second attack is less difficult. We need here to exclude hysteria, extreme excitability of the nervous system (allowing of a late eclamptic or reflex seizure), uræmia, and syphilis inherited or acquired. Yet in the syphilitic cases the attacks may rightfully be designated epileptic, as contradistinguished from spasms caused by reflex action. Even at the menstrual period, an epileptiform attack occurring in a girl is probably the beginning of epilepsy. In adults, uræmia is the factor to be most carefully eliminated, especially if the first seizure has been a *status epilepticus* with long-continued stupor. In uræmia the spasm is less distinctly tonic at first, biting of the tongue is rare, small pupils are the rule. The temperature may be high as in epilepsy, and it should not be forgotten that uræmic spasm, like Jacksonian epilepsy, may be hemiplegic or one-sided. Examination of the urine is not as valuable a test as might be expected *a priori*, for albumen may appear in the urine as the result of a truly epileptic attack, and on the other hand, uræmia so-called, may be present without albuminuria and only few casts. The examinations of the urine should be frequently repeated (three or four specimens

in each day) during the two or three weeks succeeding the seizure. In this way evidence of chronic interstitial nephritis may be obtained. There are, I believe, rare cases in which eclamptic attacks precede the appearance of objective signs of renal disease in the urine, several such attacks recurring at long intervals before the diagnosis of cirrhosis of the kidney can be made. As regards syphilis, it is of the utmost importance to recognize it, but its discovery does not relieve us of the obligation of beginning a mild anti-epileptic treatment, as well as an anti-syphilitic treatment, after the first seizure.

In case of doubt in an adolescent or adult, as to the real significance of a first or second epileptiform attack (that is, as to whether it is truly epileptic or symptomatic), I think the best course is to begin a moderate, carefully watched bromide treatment, and continue it a long time. If properly managed in a subject not unusually affected by the bromides, I firmly believe that such a course will do no harm, and may, probably will, cure the beginning epilepsy.²⁰ If we hesitate, and postpone an anti-epileptic treatment, how great a responsibility is thrown upon us. Each recurring attack diminishes the prospects of a cure in an enormous ratio I believe, and after three, six, eight convulsions, spread out it may be over a period of two or three years, the case is probably incurable. I would repeat what I said at the beginning of this section, that if there is any disease the curability of which depends on its recognition at the earliest possible moment, that disease is epilepsy. Lastly, if it appears that we have given a long-continued bromide treatment to a non-epileptic person, after an eclamptic attack, no special harm has been done. It is a serious dilemma for the physician, but to my mind there is no doubt as to which course should be chosen for the patient's welfare.

(2) The non-recognition of *petit-mal*. This is a very frequent error on the part of parents and of physicians as well. The "spells" are so slight that they surely amount to nothing; they come from the stomach; they will pass off when the child is older, etc. Numberless reasons are given for the neglect of these

²⁰ The fear of dementia from bromism is prevalent in the minds of physicians and laymen. It is based on the results of reckless, unsystematic giving of bromides, producing toxic effects. I have repeatedly received patients in this condition, and still having attacks, and have been able, by *care*, to reduce the doses of bromide, restore their intelligence, and diminish the number of attacks still further. It should be remembered, and impressed on parents that it is the epilepsy which causes dementia, as was perfectly recognized by authors writing before bromides were used.

cases. Another fatal misconception is that such "dizzy spells" will cease when menstruation is established; a most absurd notion. If you will pardon a digression, I will speak here of the dangerous error, popularly universal and indulged in by too many physicians, that chorea and epilepsy are benefited by menstruation and by marriage. In my experience nothing could be more erroneous and dangerous, especially as regards marriage. Neuroses are aggravated by both these events, and we should use all our influence, I believe, to prevent the marriage of epileptics. I have known marriage to be *prescribed* for epilepsy to both male and female subjects, in all cases with very bad results, physically and socially. Such practice is deserving of the strongest condemnation.

The question of the marriage of epileptics, apart from this fanciful therapeutic influence, is one often presented, and each case requires separate judgment. In general I am opposed to the marriage of any epileptic, yet exceptions occur. For instance, if the patient be a woman, and the prospective husband fully understands the circumstances, if she is not herself very degenerate or come of degenerate stock, if her attacks are under control, and if the man be not neurotic or degenerate, I would consent to the marriage. I have studied the children issued of two or three such marriages, and see no reason to regret my action. In the case of a male epileptic the question is more serious. He is supposed to be the bread-winner and protector of the wife and family, consequently very slight remains of the dread disease should positively prohibit marriage. Given the case of a healthy young epileptic, not degenerate, not of degenerate stock, who has had no attack for four years, under treatment, I might give consent, but only on condition that the *fiancée* should know all particulars, and fully appreciate the possibility of a return of the disease, and of disability of her husband. It is, gentlemen, a question which comes very close to our conscience, and we should not allow any sentimental notion to bias our judgment. A newly-married man or woman may honestly swear to care for an infirm (epileptic) wife or husband, yet after a few years of contact with the repulsive symptoms of the disease, especially the moral perversion which characterizes it (so few epileptics can be good, true or kind), it becomes humanly impossible to fulfil the promises, and cruel wrong results, besides the possible tainting of the offspring.

To return to *petit-mal*, the points for its diagnosis

have been given above. I beg you not to underestimate these slight symptoms, but to institute a thorough treatment as soon as possible. A remarkable fact, noted by many observers, is that *petit-mal* does not yield to bromide treatment as readily as *grand-mal*. Indeed, many cases show no improvement even when bromism is well marked, and I must admit that we, as yet, know of no indications for the use of other remedies. It is a hap-hazard, experimental treatment. Some cases yield, as if by magic, to strychnia, others to atropia, some to ergotin and digitalis. I have exhausted my *materia medica* in some few cases without materially reducing the number of attacks (often many each day). It is always a painful surprise for parents when you tell them, as you should, that these slight "spells," hardly worth mentioning, are much more difficult to control than convulsions, and that they are just as likely, if not more so, to affect the child's intellect.

For many of the minor points in the diagnosis of *petit- and grand-mal* in children I would again refer you to my former paper.²¹ As an encouragement I might cite here a case which has been under my care for the past two years; slight dream-like *petit-mal* in an adult, no convulsions. With a moderate dose of bromide of sodium (three grammes) on rising, and *cannabis indica* 0.02 three times a day, tonics and hygiene, an interval of nearly a year has been obtained. The patient had had for several years attacks almost every day. Dementia was slightly but distinctly marked when I first saw him, but his mental action has become better and easier in the last year, in spite of great business responsibilities.

Pardon me, if, before such an audience, I have touched upon so many trivial points. My experience as a specialist has shown me that the diseases treated of in this paper are not recognized as early as they should be and can be, if the general practitioner will give *attention and time* to the study of his cases by the analytical method. Especially in epilepsy is an early diagnosis urgently demanded, because it is the most curable of the diseases mentioned. Pray believe me when I tell you that you will never regret having made an early diagnosis of tabes, dementia paralytica, spinal disease, cerebral tumor and epilepsy; whereas we all look back with self-reproachful feelings to having failed to recognize these affections early, and to institute proper treatment, medicinal or hygienic.

²¹ Opera Minora, p. 540, et seq.

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